

**WHAT IS CLAIMED IS:**

1. A method for the treatment of overproduction of mucin in a mammal, comprising:

administering an inhibitor of p38 MAP kinase to the mammal in an amount sufficient to reduce mucin production

2. The method of Claim 1 wherein the overproduction of mucin is caused by an otitis media (OM) infection or chronic obstructive pulmonary disease (COPD).

3. The method of Claim 2 wherein the OM or COPD is caused by nontypeable *Haemophilus influenzae* (NTHi).

4. The method of Claim 1 wherein said inhibitor of p38 MAP kinase is a chemical inhibitor selected from the group consisting of: pyridimylimidzol SB203580, SB202190, SB220025, SC68376, SKF-86002, a dominant-negative mutant of p38 $\alpha$ , and a dominant-negative mutant of p38 $\beta$ .

5. The method of Claim 1 wherein the inhibitor of p38 MAP kinase is an antisense oligonucleotide.

6. The method of Claim 1 wherein the inhibitor of p38 MAP kinase is a vector which expresses a protein or polypeptide which inhibits p38 MAP kinase.

7. The method of Claim 1 wherein the method of administration is selected from the group consisting of: inhalation, ear drops, transtympanically, intramuscularly, intravenously, and by mouth.

8. A method for the identification of regulators of mucin production, comprising:

providing a reporter vector containing the MUC5AC or p38 MAP kinase promoter;

contacting the reporter vector with a potential regulator; and identifying the up-or down-regulation of the reporter gene.

9. The method of Claim 8, wherein said potential regulator is selected from the group consisting of: a polypeptide, a polynucleotide, and a small molecule.

10. The method of Claim 8, wherein said potential regulator is a mixture of proteins from a cell.

11. The method of Claim 8, wherein said potential regulator is an antisense polynucleotide.
12. The method of Claim 8, wherein said potential regulator is a library of small molecules.
- 5 13. A method for the treatment of overproduction of mucin in a mammal, comprising:  
administering an activator of PI-3 kinase to the mammal in an amount sufficient to reduce mucin production.
- 10 14. The method of Claim 13 wherein the overproduction of mucin is caused by a disease selected from the group consisting of: Otitis media, chronic obstructive pulmonary disease, asthma, and cystic fibrosis.
- 15 15. The method of Claim 14, wherein said overproduction of mucin is caused by otitis media (OM) infection or chronic obstructive pulmonary disease (COPD).
- 16 16. The method of Claim 14 wherein the OM or COPD is cause by nontypeable *Haemophilus influenzae* (NTHi).
- 17 17. The method of Claim 13 wherein said activator of PI-3 kinase is a protein selected from the group consisting of: a dominant negative mutant of PI-3 kinase, a constitutively active form of p110 (p110-CAAX), wildtype Akt.
- 20 18. The method of Claim 13 wherein the inhibitor of p38 MAP kinase is an antisense oligonucleotide.
19. The method of Claim 13 wherein the inhibitor of PI-3 kinase is a vector which expresses a protein or polypeptide which activates PI-3 kinase.
20. The method of Claim 13 wherein the method of administration is selected from the group consisting of: inhalation, ear drops transtympanically, intramuscularly, intravenously, and by mouth.
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